

REMARKS

Disposition of Claims

Claims 1-4, 6, 8, 17-18 are being examined. Claims 5 and 7 have been cancelled. Claims 9-16 have been withdrawn as directed to a non-elected invention.

Detailed Action

Summary of the Invention

The present invention relates to the finding by the present inventors that a cell surface antigen of Chlamydia has mannose-6-phosphate ("Man-6-P"). Based upon this finding, the inventors hypothesized that Chlamydia infects target cells by engaging the Man-6-P receptor found on the target cell. The inventors also noted that the Man-6-P receptor is multi-functional (it has one binding site for phosphomannosyl residues and another for IGF-2), and that insulin-growth factor 2 ("IGF-2") enhances the susceptibility of human endothelial cells to infection by Chlamydia pneumoniae (IGF-2 is a mammalian hormone produced by human monocytes such as macrophages).

The present invention further relates to inhibiting infection of cells by Chlamydia by interfering with the interaction of the Man-6-P, found on the Chlamydia cell surface antigen, with the Man-6-P receptor, found on the target cell. The inventors discovered that the interaction can be interfered with by molecules that interact with the Man-6-P on the Chlamydia structure, the Man-6-P receptor on the target cell, or the insulin-like growth factor in the environs of the target cell. Thus, the present invention provides a composition comprising:

- (a) A Chlamydia infection inhibiting amount of a molecule that interacts with Man-6-P, Man-6-P receptor, and/or insulin-like growth factor; and
- (b) A pharmaceutically acceptable carrier, diluent or excipient.

Rejections Maintained

- (1) Claims 1 and 8 remain rejected under 35 U.S.C. § 102(b) as being anticipated by *Kuo et al.* (J. Clin. Invest. Vol. 98(12) pp. 2813-2818).

The Examiner's position appears to be that *Kuo et al.* show that HeLa cells treated with high mannose type oligosaccharide inhibited infectivity of Chlamydia MOMP and that the HeLa cells pretreated with oligosaccharides would interact with mannose-6-phosphate. Therefore, the composition comprising pretreated HeLa cells with oligosaccharides is a Chlamydia infection inhibiting molecule that interacts with mannose-6-phosphate, as required claims 1 and 8.

The Examiner's position is traversed, respectfully, because there is no evidence that HeLa cells pretreated with oligosaccharides interact with mannose-6-phosphate. Furthermore, a pretreated HeLa cell is not a "molecule" as required by the claims. That is, the claims do not recite a composition that inhibits infectivity and interacts with mannose-6-phosphate, but rather a molecule that inhibits infectivity and interacts with mannose-6-phosphate.

Therefore, the rejection is improper and should be removed.

- (2) Claims 1-4, 6 and 8 remain rejected under 35 U.S.C. § 102(b) as being anticipated by *Ooij et al.* (Infect. Immun. 1997 Vol. 65(2) pp. 758-766).

The Examiner appears to be saying that the disclosed monoclonal antibody to mannose-6-phosphate receptor is a molecule that inhibits Chlamydia infection, because in infected cells,

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Chlamydial vacuoles were shown to bind to the antibodies and Chlamydial vacuoles have been shown to be involved in replication.

The Examiner's position is traversed, respectfully. The rejection is being made in hindsight. That is, the Examiner refers to applicants' specification and discovery that some molecules that react with mannose-6-phosphate receptor can also inhibit Chlamydia infection. The Examiner then asserts that the disclosed antibody, which interacts with mannose-6-phosphate receptor, must also be able to inhibit Chlamydia infection. However, there is no support for this assertion. Being "involved in replication" is not sufficient to conclude that a molecule can inhibit infection. This assertion can only be supported by reference to the present specification. Such hindsight use of applicants' specification is improper. Therefore, the rejection must fail and should be removed.

(3) Claims 1-8 remain rejected under 35 U.S.C. § 102(a) as being anticipated by *Lin et al.* The Examiner states that an executed Declaration to remove the reference has not been submitted.

Submitted herewith is an executed Declaration by the inventors stating that the subject matter of *Lin et al.* was derived from the claimed invention.

Accordingly, the rejection is overcome and should be removed.

New Rejections

Claim Rejections - 35 U.S.C. § 112

Claim 8 is rejected as being indefinite in the recitation of "molecule comprises mannose-6-phosphate." The Examiner states that it is not clear how an infection inhibiting molecule that comprises mannose-6-phosphate interacts with mannose-6-phosphate.

This rejection has been overcome by amending claim 1 to recite that the molecule interacts with "one or both of" mannose-6-phosphate and mannose-6-phosphate receptor.

Accordingly, the rejection should be removed.

Claim Rejections - 35 U.S.C. § 102(b)

(1) Claims 17 and 18 are newly rejected under 35 U.S.C. § 102(b) as being anticipated by *Ooij et al.*, 1997.

The Examiner appears to be saying that the antibody to mannose-6-phosphate receptor would also react with IGF in infected HeLa cells, and thereby inhibit further infection. The Examiner then states that the composition of an antibody treated infected HeLa cell is a molecule that inhibits Chlamydia infection.

The Examiner's position is not correct because a "molecule" as recited in the claims does not include within its scope a composition comprising cells. Furthermore, under the Examiner's interpretation of the reference, the "molecule" is defined inconsistently. That is, the Examiner asserts that the antibody is the "molecule" that interacts with insulin-like growth factor, whereas antibody bound to the cell is the "molecule" that is present in an infection inhibiting amount.

Therefore, the rejection is based upon a misinterpretation of the technology and/or the claims and should be removed.

(2) Claims 17 and 18 are rejected under 35 U.S.C. § 102(b) as being anticipated by *Peterson et al.* 1998 (Infect. Immun. Vol. 66(8) pp 3848-3855).

The Examiner appears to be saying that monoclonal antibody CP-33 disclosed by *Peterson et al.* specifically binds to Chlamydia and neutralizes infection by Chlamydia

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pneumoniae and, therefore, inherently or necessarily the antibody is interacting with insulin-like-growth factor on Chlamydia.

The Examiner's rejection appears to be stating that insulin-like growth factor is found on Chlamydia. This is not possible. Insulin-like growth factor is a mammalian hormone found in serum, not on bacteria. Therefore, the antibody disclosed by *Peterson, et al.* could not possibly be interacting with insulin-like growth factor. As a result, the CP-33 antibody does not meet the limitation of the claims and the rejection is improper and should be removed.

In view of the above, reconsideration and allowance of this application are now believed to be in order, and such actions are hereby solicited. If any points remain in issue which the Examiner feels may be best resolved through a personal or telephone interview, the Examiner is kindly requested to contact the undersigned at the telephone number listed below.

The USPTO is directed and authorized to charge all required fees, except for the Issue Fee and the Publication Fee, to Deposit Account No. 19-4880. Please also credit any overpayments to said Deposit Account.

Respectfully submitted,



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